Paroxysmal dyskinesia presenting feature of hyperthyroidism in an elderly: A case report

Ayush Dubey[1], Sunil Kumar[2,*], Anuj Chaturvedi[3], Akshay Daphal[4]

[1,3,4]Post graduate Resident, [2]Professor & HOD, [1-4]Dept. of Medicine, Jawaharlal Nehru Medical College, Datta Meghe Institute of Medical Sciences, Wardha, Maharashtra, India

***Corresponding Author:**
Email: sunilkumarmed@gmail.com

Abstract
Paroxysmal dyskinesia (PD) as a first manifestation of hyperthyroidism is an extremely rare entity. We here report a case pf 65-year-old female presented with the features of PD, whose investigations revealed hyperthyroidism. Her dyskinesia responded with the anti-thyroid drug carbimazole.

Keywords: Dyskinesia, Hyperthyroidism, Elderly, Carbimazole.

Introduction
Paroxysmal dyskinesia (PD) is an unusual dyskinesia characterized by brief unilateral or bilateral chorea, choreoathetosis, ballism, or dystonic postures without any changes in consciousness. PD is often precipitated by voluntary movement and it is often seen in multiple sclerosis, ischemic stroke, head injury, drug abuse, diabetes mellitus, or hypoparathyroidism. There are only three case reports describing PD related to hyperthyroidism, in the literature, none till date have been reported in elderly.[1-3] We report a case of PD in a 65 year old hyperthyroid elderly patient in whom the involuntary movements disappeared after antithyroid drugs.

Case Report
A 65-year-old female presented in medicine out patient department with abnormal movement of jaw since one month. Her consciousness remained clear throughout the attack. The abnormal movements were transient in nature, lasting for 2-3minutes, mostly triggered by voluntary activities like taking meal or drinking water. These attacks would occur several times in a day with variable inter-attack interval. She never experienced heat intolerance, palpitation, diarrhea, weight loss or tremors.

Physical examination was non-contributory except palpable swelling in the neck. There was no bruit on auscultation over the swelling. Detailed neurological examination was essentially normal. There was no KF ring on slit-lamp examination. Both fundi were normal. The episodes were stereotyped in the form of oromandibular dystonia or facial grimacing.

Her hemogram, including red cell morphology, blood chemistry for blood sugar, blood urea, serum creatine, serum electrolytes, and profile for liver function, lipid, serum copper, ceruloplasmin, serum calcium, phosphorus, and parathormone level were normal. Ultrasonography of neck swelling revealed multinodular goiter and on fine neddle aspiration cytology it was colloid goitre. Serum free triiodothyronine was 4.6 pg/ml (normal range 2.3-4.2 pg/ml), free thyroxine 1.3 ng/dl (normal range 0.89-1.8 ng/dl), thyroid-stimulating hormone was 0.01 Iu/ml (normal range 0.35-5.5 Iu/ml). Computerised tomography scan of brain was normal. Electro Encephalography did not show any epileptiform discharge.

She was put on neomercazole 20 mg in divided doses and trihexyphenidyl 2 mg twice a day. The patient did not show any response initially. However, she responded two weeks after the initiation of the medication and became euthyroid after about one month. The trihexyphenidyl was gradually withdrawn after one week and she never experienced the attacks after its withdrawal.

Discussion
Hyperthyroidism rarely presents with movement disorder like, dystonia, hemiballismus, chorea or choreoathetosis, which usually resolves with the use of anti-thyroid medications.[4-6] The exact mechanism of how thyroid hormones induce paroxysmal dyskinesia is not clear. Functional hypersensitivity of dopaminergic receptors in the striatum may be responsible for some movement disorder like hyperthyroid chorea.[1]

In our patient hyperthyroidism first presented as PD. PD are divided in into three main categories: paroxysmal kinesigenic dyskinesia, paroxysmal non-kinesigenic dyskinesia, and paroxysmal exercise-induced dyskinesia.[4,5] Positron emission tomographic studies are usually normal in these patients. However, decreased striatal glucose metabolism was observed in most patients with chorea, suggesting a pathogenetic mechanism different from that of PD. One case report presented a patient with a 7-year history of atypical PD with intracerebral calcifications secondary to hyperthyroidism.[6] This case had no structural lesion on neuroimaging, it can be postulated that hyperthyroidism-related PD is more likely the result of

a metabolic disturbance of the basal ganglia circuits rather than a permanent and irreversible change.

Due to their atypical and unusual clinical presentations, rare paroxysmal movement disorders have frequently been labelled and misdiagnosed as a psychogenic movement disorder.[5] This may also be due to a lack of awareness among physicians. Our patient did not have any psychiatric symptoms. Thus, the present case is unique with PD as the presenting manifestation of hyperthyroidism.

References

1. Puri V, Chaudhry N. Paroxysmal kinesigenic dyskinesia manifestation of hyperthyroidism. *Neurol India* 2004;52:102-3.
2. Khan WU, Staios G, Rana AQ. Paroxysmal kinesigenic dyskinesia in a mother and daughter. *Acta Neurol Belg* 2010;110:201–2.
3. Kailash PB. Paraxysmal dyskinesias. *Mov Disord* 2011;26:1157–65.
4. Van Rootselaar AF, Schade van Westrum S, Velis DN, Tijssen MA. The paroxysmal dyskinesias. *Pract Neurol* 2009;9:102–9.
5. Abhishek, Anand KS, Premsagar IC. Sporadic paroxysmal non-kinesigenic dyskinesia: A frequently-misdiagnosed movement disorder. *Singapore Med J* 2007;48:e250–52.
6. Demirkiran M, Jankovic J. Paroxysmal dyskinesias: Clinical features and classification. *Ann Neurol* 1995;38:571–79.

How to cite this article: Dubey A, Kumar S, Chaturvedi A, Daphal A. Paroxysmal dyskinesia presenting feature of hyperthyroidism in an elderly: A case report. Ann Geriatrics Educ Med Sci. 2018;5(2):77-78.